THE AETIOLOGY OF RHEUMATIC FEVER WITH SPECIAL REFERENCE TO IT AS A SEQUELA TO SORE THROAT.

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I.

Brief Historical Survey.

Within the last quarter of a century the Aetiology of Acute Rheumatism has taken up a new position, and the bacterial origin and infectious nature of the disease are now almost universally accepted.

In order to bring out the change in attitude regarding the cause of Rheumatic Fever which has taken place during this period, an historical survey will be given.

The chemical theories held favour for a period, uric acid and lactic acid being put down as the causal agents. Prout, who originated the lactic acid theory, was supported by the experiments of Richardson and Rauch. who produced pericardial and endocardial lesions with lactic acid. The uric acid theory, however, of Latham and Haig has not had any very convincing experimental evidence to support it.

The theory that cold was the causal factor was brought forward by Cullen, who thought that the joints, on account of their thin covering, were affected by cold, and that the condition originating in the joints spread to other systems. Mitchell, on the other hand, was of opinion that cold irritated the sensory nerve fibres and started a primary focus of Rheumatism in the spinal cord, from which it passed by the nerves to the various organs, which were in time affected with the rheumatic condition.

With the introduction of the microbic theory, other theories as to cause have gone to the wall. In Rheumatic Fever, or in what clinically appears to be Acute Rheumatism, various organisms at various times have been discovered by different observers. Guthmann in 1890 found the staphylococcus pyogenes flavus in a case of Acute Rheumatism complicated by a renal abscess. The organism was isolated from the abscess and pericardium, but not from the joints. Guthmann himself, however, was doubtful as to this organism being the causal one of Acute Rheumatism. Sahli, about the same time, isolated

the staphylococcus pyogenes citreus from the synovial membrane of the joints and from the pericardial exudate in a case of Acute Rheumatism. Netler demonstrated a staphylococcus in a case of Acute Rheumatism, which had, however, attacked a patient who had just previously suffered from Acute Otitis Media. In 1893, Lanz published a paper on Suppurative Polyarthritis. In it he stated that he had isolated a bacillus from a cerebral abscess, which, when injected intravenously into rabbits, produced suppurative polyarthritis, the organism being again isolated from the joints of these animals. He christened his organism Bacillus pyogenes foetidus liquefaciens. About the same time, Maragliano found staphylococci and diplococci in a case of Rheumatic Fever, which, however, ultimately became Septicaemia. Singer isolated the staphylococcus pyogenes aureus from the urine of patients suffering from Acute Rheumatism. In 1894, Dana obtained a diplococcus from the meninges of the brain and spinal cord of a case of Chorea.

The findings of these observers pointed to the fact that, although Acute Rheumatism was due to an organism, it was in no way due to a specific one, but that organisms that produce pus and suppurative

conditions as a whole might under certain conditions produce Acute Rheumatism, and that the condition is simply one of Septicaemia.

Sacaze suggested that the site of infection was some external wound, whereas Lubauch and Fowler point to the tonsils as the initial site of infection. Achalme discovered a bacillus in a case of Rheumatic Fever, and between 1891 and 1897 published his views in a series of papers. His bacillus was isolated from the blood of patients after death. It was described as an anaerobic organism, staining with aniline dyes, and in size and shape resembling the anthrax bacillus. When injected into animals, Rheumatic Fever as found in man was not however produced, but a form of septicaemia. This bacillus was also found by Thiroloix, Triboulet and Bettencourt, the last-named producing Rheumatic Fever in rabbits with it. The investigations of Achalme, Thiroloix, Triboulet and Bettencourt point to Acute Rheumatism being due to the invasion of a definite bacillus.

Triboulet and Coyon, however, discovered a diplococcus occurring coincidently with Achalme's bacillus, and made the observation that severe and complicated

cases of Acute Rheumatism owe their origin to the combined action of these two organisms, whereas simple cases are due to the diplococcus alone. Dr.Alfred Mantle about 1886 had demonstrated a diplococcus which he obtained from the blood and serum withdrawn from the knee joint of patients suffering from Acute Rheumatism. He cultivated these, and experimented upon monkeys with injections of his cultures, without, however, producing symptoms of Rheumatism in them.

Wassermann in 1899 also drew attention to a diplococcus which he had discovered during his investigations on Rheumatism.

Poynton and Payne, in 1900, published in "The Lancet" an article in which they stated that they had isolated a diplococcus from cases of Acute Rheumatism. This organism, they asserted, was the cause, if not the only one, of that condition. This diplococcus is identical, they state, with that discovered by Wassermann and Triboulet. They are micrococci 0.5 μ in size, arranged in pairs, growing in chains in liquid media, and resembling staphylococci when grown in solid media. They can be cultivated from the tissues in an acid medium of broth and bouillon, and can be grown either anaerobically or

aerobically. After isolation, they can be grown on blood agar at body temperature. They stain with aniline dyes and are gram positive.

The investigations of Poynton and Paine on the Aetiology of Acute Rheumatism are by far the most important which have been carried out in this country. I shall therefore give in some little detail an account of their labours in this direction.

Drs. Poynton and Paine had for some considerable time been induced to infer from their clinical observations on Rheumatism that it was a specific disease, due to a special infection. From about 1898 till the time of their discovering the diplococcus in 1900, they published numerous papers on the subject. These papers deal with the acute dilatation of the heart which occurs in children suffering from Acute Rheumatism and Chorea. In the first paper on this subject, the authors state their observations on the size, strength and sounds of the heart in healthy children between the ages of seven and fourteen. Following these, are observations on the heart of children suffering from Rheumatism and Chorea. These articles demonstrate that a toxic agent is present, which, acting on the heart muscle, weakens it, causing the heart to dilate under the normal blood pressure; and

that this dilatation is quite apart from enlargement of the heart due to pericarditis, or to dilatation or hypertrophy due to endocarditis.

Other papers deal further with this subject. Cases are described in which there was a coincident pericarditis. The post-mortem changes in the heart wall are described, and a comparison is drawn with the changes brought about by other morbid conditions.

In all these papers, the infectious nature of Rheumatic Fever is kept prominently before the reader.
In 1899, Poynton and Paine commenced to investigate the bacteriology of Rheumatism by an attempt to establish the existence of the anthrax-like bacillus of Achalme Disappointment and failure, however, confronted them in this respect, and they were quite unable to prove the existence of any such organism. They did succeed, however, in discovering the diplococcus, which they now hold to be the causal agent in Acute Rheumatism. Great difficulty was at first experienced in demonstrating these organisms in the tissues, but at last this was overcome, and they were successfully obtained from the blood, pericardium, valves of heart, tonsils and rheumatic nodules.

II.

Mode of Infection and paths by which organism enters into the system, with special reference to the Tonsils. Special tissues affected, with a discussion on the morbid changes and brief reference to clinical signs.

There appears to be little doubt that Acute Rheumatism is the result of infection by a pathogenic organism, and that this organism is the Diplococcus Rheumaticus as demonstrated by Poynton and Paine. The patient becomes infected, the organism enters the blood-stream, in which it travels to its special sites of selection, settles down, multiplies, and forms its toxins. In all probability, the tonsils are the primary seat of infection, the diplococcus passing from these by the lymph-stream to the blood. That the tonsils are the primary site of infection receives strong support from many clinical investigators, and also from the post-mortem findings of pathologists. The rheumatic sore throat, with enlarged tonsils, of children is an extremely common occurrence in general practice, and is very frequently accompanied by cardiac dilatation and later followed by graver heart mischief. In cases where the morbid changes in the tonsils have been studied after death, they have presented the appearance of subacute

inflammation, are indurated, and on section show masses of inflammatory exudate throughout the structure of the organ. During life, the diplococcus has been isolated from the tonsils of patients suffering from Acute Rheumatism by Poynton and Paine, Meyer and other observers. Accompanying these attacks of tonsillitis in children, there is frequently a moderate degree of pyrexia, and at the same time, or at other times, the child complains of vague pains (growing pains) in the limbs, and sometimes of mild joint pains. Unfortunately, the symptoms are often so mild that the child is not confined to bed, nor is a medical man consulted. It is often only when the child at a later date is brought under the notice of the doctor, that, on account of the cardiac conditions found, enquiry is made into the case and a history of sore throat elicited. I have come across these cases frequently in my dispensary and private practice, where the child has been now and again slightly ill with sore throat and mild, vague joint pains, and at the time of my examination has had an organic mitral lesion. Most of these children also on examination have had chronically enlarged tonsils. The tonsils, therefore, are an important portal for the entrance of the rheumatic

organism, whence it travels to the various tissues for which it has a special affinity. These tissues are the synovial membranes of joints, the endocardium of the heart(especially that portion which is associated with the valve cusps), the pleura of the lungs and the pericardium. The myocardium also is attacked -- more often, however, by the toxin than by the organisms. The peritoneum and meninges may also be attacked. In this respect, the diplococcus rheumaticus resembles the pneumococcus, gonococcus and other organisms of the group. Rheumatism is analogous to a pyaemia. The diplococcus circulates in the blood, settles down in certain tissues and organs, multiplies and produces its toxins. Garrod points out that two factors are necessary for the production of rheumatism - first the organism, and second the toxin. The organism produces the gross lesions, such as the heart lesion, whereas the toxin produces the transient joint pains and swelling, the cardiac dilatation, the nervous symptoms, etc. In adults, the toxin would appear to be accountable for the major portion of the manifestations, whereas in children they would appear to be due to the local growth of the organism.

Having briefly discussed the method by which the organism of Rheumatism gains access to the blood-stream of the infected individual, and having noted the part played by the tonsils in admitting the enemy, it would be as well perhaps to mention the organs of the body which are most commonly selected for the pathogenic attentions of the causal organism of Rheumatism, and to describe the morbid changes which take place in them. As acute Rheumatism, at least in adults, is usually described as being characterised by a polyarthritis, the joint changes will first of all be discussed. Clinically, the joints affected are moderately swollen and very painful on the least movement. The synovial membrane becomes congested and swollen, and an effusion of an albuminous fluid takes place. This fluid is clear, or may be turbid. It rarely, if ever, becomes purulent, however. The ligaments of the joint become swollen, and slight erosion of the cartilages may occur. The affection of the joints has a metastatic character, first one joint, then another, being affected, the first probably being practically better before the other is attacked. Several joints may be affected at the same time. The joint condition usually, unlike the

heart, completely recovers, this possibly being due to the fact that the joint can be put at and kept at complete rest, whereas the heart cannot. Garrod, however, suggests that the joint affection is due to the toxin, whereas the heart one is caused by the organism itself, thus in a measure accounting for the fleeting character of the joint affection and its non-permanency, as against the constant and permanent lesion of the heart. The Rheumatic nodule is an interesting feature with regard to rheumatic infection. The organism can now be isolated from these nodules, and their number and constancy markedly influence the prognosis in any given case of Rheumatism. They are, however, by no means constant or even very frequent features in every-day Rheumatic Fever. Since returning to civil practice, I have not been fortunate in attending cases which included these nodules in their manifestations. Poynton's investigations on the Rheumatic nodule tend to show its great importance, and to demonstrate the similarity which exists between the rheumatic changes which take place in these nodules and those which occur in rheumatic endocarditis and pericarditis. These rheumatic nodules, when they occur, are small bodies, varying in size from a pin-point to a good-sized pea. Those of

the former size cannot, of course, be palpated and demonstrated during life, but are to be found on post-mortem examination. The nodules, when present, are usually to be found in one or other or several of the following regions: about the elbows, knees, ankles, knuckles, posterior spinous processes of the vertebrae and occipital curved lines, as well as over prominent tendons. They are subcutaneous, and are situated over the bony prominences in the localities above mentioned. They are not as a rule attached to bone, but may be so in regions where the subcutaneous tissue comes into very close relationship with the periosteum, as, for example, in the skull. As a rule, however, they are freely movable. They are generally symmetrically placed on the two sides of the body and tend to come out in crops, new ones appearing before the old ones have quite disappeared. Each nodule usually lasts several months and then gradually disappears. It may, however, only last a day or two. Histologically, when old, these nodules are seen to be made up for the most part of fibrous tissue. When recent, however, they show the fibrous tissue only around the periphery. This peripheral tissue has large, tortuous and distended

blood-vessels penetrating it. Nearer the centre are numerous cellular elements; whereas in the centre is a homogeneous fibrinous material, arranged in layers, and having fluid in the spaces between its strands, in which the diplococcus is to be found. Dr. Poynton points out the very close resemblance there is between a recent rheumatic nodule and a recent peri- or endocarditis, and from his study of the two latter conditions explains the rapid appearance and disappearance of the nodule. In Rheumatic peri- and endocarditis, he states, there is firstly vascular dilatation, then fibrinous exudation, later cellular infiltration, and lastly fibrosis. In the case of the nodule, absorption may take place if the Rheumatic process is rapid at the fibrinous stage, when the nodule in consequence vanishes.

The Rheumatic nodule has been discussed somewhat at length, on account of its many interesting points, and especially because of the relationship of its changes to those of the Rheumatically affected heart.

The heart is perhaps the most important organ to be affected by the Rheumatic condition, and it is certainly the one which is most frequently permanently

damaged by the process. The pericardium, myocardium and endocardium may all be affected, individually or together. The myocardium is practically always involved, if the condition is at all active. Permanent damage does not, however, so readily follow as in rheumatic infection of the endocardium. The myocardium is affected chiefly by the toxin, which may affect it in two ways, either by causing loss of tone of the muscle and consequent dilatation, or by causing a diminution and weakening of its contractile power. In rare cases the conductivity of the cells of the primitive heart muscle may be interfered with and cardiac irregularity result. The endocardium, especially in children, if the attack be at all severe, is nearly always involved, and chronic changes are very apt to follow. The endocarditis may be the first clinical evidence of the disease. Clinically, it is at times difficult to determine at the outset whether the physical signs are due to endocarditis or to the associated myocarditis. The morbid changes are present most frequently in the valves, although other parts of the endocardium may be attacked. Infection possibly takes place from the deposition of

the organism from the circulating blood, and this might account for the more frequent involvement of the valves. Poynton and Paine, however, state that the organisms are conveyed to the base of the valve by the capillaries, and thence by the nutrient channels to the subendothelial tissue, where they cause cell proliferation, cloudiness, swelling and thickening of the valvular endocardium. Small granulations are then formed, fibrin is laid down, and a vegetation thus formed. After a time, these vegetations, which are usually situated at a short distance from the free margin of the valves on their proximal surfaces, are converted into fibrous tissue. As stated above, it is sometimes difficult to say at the outset whether the signs are or are not simply due to the associated myocarditis. The increased pyrexia without any very definite reasons for its presence, and the indistinct, ill-defined first sound, with a reduplication of the second sound in the mitral area, associated with a displaced apex beat and increased area of cardiac dulness, are, however, highly suspicious of endocardial involvement, Later, a typical organic bruit may develop. Of the cusps attacked, those of the mitral and aortic valves are most frequently selected. The heart need not, however, be permanently

damaged by an attack of Acute Rheumatism, and may withstand an attack, or even recover after being the seat of infection. This is more frequent in the Rheumatism of adults than in that of children; nor does the gravity of the lesion depend in any way upon the severity of the general symptoms in the attack. In fact, a mild attack may cause a much graver cardiac lesion than a severe one. The repeated attacks of childhood, where general rheumatic symptoms are wanting, are the ones which play havoc with the heart valves. These slight feverish attacks, slight sore throat and tonsillitis, with vague joint pains, in children, are the ones which are accountable for such a very large percentage of the cardiac valvular lesions found in later years. In the very large number of young adult hearts with organic lesions which I have examined recently, fully 75% have had a history of Rheumatism, and I am convinced that,if one had been able to elicit a satisfactory medical history from the remainder, this percentage would have been largely increased. The malignant form of Acute Endocarditis may follow as a result of Rheumatic infection. Fortunately, however, this is a somewhat rare occurrence. We have seen that the acute endocarditis may be completely recovered from,

or, on the other hand, may, and very frequently does, develop into chronic endocarditis, affecting either the mitral or aortic valves, separately or together. The lesion may be a stenosis, an incompetence, or a combination of these two. The morbid Anatomical conditions of chronic endocarditis are just a stage further in advance of those which occur in the acute form - the formation of fibrous tissue, the thickening, shortening and stiffening of the cusps, leading to incompetence, and the adhesions of their margins, leading to stenosis. Calcification frequently takes place later in these fibrotic cusps, the clinical signs being, of course, the various bruits and cardiac enlargement, accompanied later by the other signs of heart disease as compensation fails. Acute pericarditis is not of such common occurrence as endocarditis. When it occurs, it is frequently associated with the latter condition, and is always accompanied by myocarditis of a more or less marked degree. In fact, the prognosis depends to a large extent upon the involvement of the myocardium. The condition does not as a rule develop at the outset of an attack of Rheumatism, but frequently comes on after the other symptoms have somewhat abated.

There is very marked constitutional disturbance, with as a rule severe pains in the region of the heart, accompanied by breathlessness and rapid irregular pulse. There is a to and fro friction rub and enlargement of the area of dulness, due in a great measure to dilatation from myocardial changes, as fluid, when later it appears, is very limited in amount, and not of nearly sufficient quantity to account for the great increase in the area of dulness. The morbid changes consist in the exudation of lymph between the parietal and visceral layers, giving a shaggy bread and butter appearance to them. The exudation of fluid, as stated above, is not nearly so abundant as might be expected. This is as a rule rapidly absorbed. Adhesions, to a more or less degree, are always a resultant of acute pericarditis. There may also be adhesions between the parietal layer of pericardium and a neighbouring viscus, resulting in great impairment of the heart's function.

The relationship between Chorea and Rheumatism has for a considerable period been remarked upon by numerous observers, the heart conditions which occur in both Chorea and Rheumatism being practically identical. In a very large number of cases, as I shall

point out later by giving figures, there is a distinct history of Rheumatism, while in other cases the rheumatic symptoms appear after the Chorea. In other cases, again, there is only a rheumatic family history.

Various theories have been put forward from time to time with regard to the causes of Chorea. Sturges declares that it is of a functional nature, the motor cells being affected. The embolic theory of Kirk seems to get nearer the mark, in so far that it can be backed up by pathological findings. The association of endocarditis, leading to vegetations, which become detached and, travelling to the brain, cause emboli in the vessels thereof, is most practical in the cases where vegetations are to be found. But where the latter are wanting on post-mortem examination, the presence of emboli requires some other explanation. That explanation of Poynton and Paine in which they state that Chorea is a nervous manifestation of Acute Rheumatism, and that the lesions found after death are due to the toxins of Rheumatic Fever, seems to meet the requirements of the case. This, they state, is probably produced locally, but may be part of the general infection, the toxins reaching the

brain by the circulating blood and lymphatic system. They have demonstrated also the diplococcus rheumaticus in the pia mater and brain substance, and have also produced ~~chronic~~ choric symptoms by inoculation of the organism into rabbits. The morbid changes to be found in chorea consist of dilatation of the blood-vessels of the brain and membranes with numerous small haemorrhages under the pia mater, and thrombosis of the vessels, especially in the cortex of the brain. Inflammatory changes are evidenced by serous exudation and small-celled infiltration, and are to be found along with the secondary degenerative changes brought about by the interference with the blood-supply.

In an article in "The Practitioner" of January 1912, Dr. Carey Coombs states that a review of over 150 cases showed that, if to those cases in which articular or cardiac Rheumatism has preceded or coincided with the Chorea attack, be added those in which Chorea has been shortly followed by polyarthritis or carditis, we find that 94% of cases of chorea in patients under 16 years of age are definitely Rheumatic. With regard to the almost constant occurrence of a heart condition in chorea,

Osler states in his article on Chorea and Choreiform Affections that in 73 post-mortem examinations of cases of chorea, 42 showed evidence of carditis. The figures given by Poynton and Paine in a paper on Researches on Rheumatism are interesting, and are as follows. They state that in 217 cases of chorea investigated by them, 122 gave evidence of heart disease and other rheumatic manifestations. In 28 more there were arthritis and muscular pains. In 22 more there was cardiac dilatation. Ten more followed a sore throat. Twenty of the remainder gave no history of any cause, but two of them later developed Acute Rheumatism. 15 were attributable to fright and shock, but in some of these no direct relation could be traced; and they were certainly rheumatic in later life. Eight were directly attributable to strain at school.

The above figures and remarks show clearly the close relationship which exists between Rheumatism and Chorea. There is another form of Chorea which might be mentioned, namely, that form which is associated with pregnancy, and in this form also the close relationship with Rheumatism has been definitely demonstrated by a host of clinicians.

The clinical manifestations of Cerebral Rheumatism present all stages from slight restlessness, headache or emotional disturbance to the actual mild or acute Chorea.

III.

Predisposing Causes and Incidence of the Disease, and a few remarks on Cases occurring in Private Practice.

We have seen that Acute Rheumatism is a Specific Infection caused by the Diplococcus Rheumaticus gaining access to the system; that some of the pathological changes brought about are due to the organism, and that others are due to its toxins. We have discussed the changes brought about in the various tissues of the body, and have seen that, whereas in older persons a typical attack affecting the joints is produced, in children these joint affections are often wanting, and when present may be so slight as to be overlooked; again, that in adults the heart is not so frequently permanently damaged as in children. One point which might be mentioned, before passing on to discuss the predisposing causes and incidence of the disease, is the distinct tendency which Rheumatism has to epidemic prevalence at irregular intervals. According to Newsholme, these epidemics occur at intervals of 3, 4 and 6 years, and vary much in their severity. In some cases several members of a family have become affected at the same time.

Rheumatic Fever occurs in temperate and humid

climates. It is very prevalent in Great Britain and Ireland, and is most prevalent during the colder months of the year. It is especially a disease of childhood, and its early diagnosis in children is of vital importance, on account of the ravages which the rheumatic organism makes upon the heart. As already mentioned, a vast majority of the chronic heart disease of adults owe their origin to attacks of Rheumatic Fever, mild or severe, in childhood. Males are affected more often than females, especially in the later years. There is a distinct hereditary tendency to Rheumatic Fever, and there is a marked rheumatic temperament. Cold and wet are predisposing causes, as also is overheating and chilling. Bad and insufficient food, unhygienic surroundings, and any factor which reduces the vitality, predisposes to an attack of Rheumatism, especially in those who have already at a previous date suffered from the disease. One attack does not produce any degree of immunity, but, like Pneumonia, makes the liability to a second or subsequent attacks more probable. Of the proportionately large number of cases of Acute Rheumatism which I evacuated from Units in

France and Belgium, fully three-quarters had a history of one or more previous attacks. Enlarged and chronically inflamed tonsils as a predisposing factor hold a prominent position, as they are the organismal entrance gates in many of the cases. The enlarged tonsils of children should be early removed, as should also those of patients who have already suffered from an attack of Rheumatism.

Of the cases of Rheumatic Fever which I have attended during the last few months, the four which I am now going to mention have all occurred in young persons.

The first case was that of a girl of fifteen years of age. The condition started with pains in the left ankle. Later, the knees were affected. The temperature was moderately elevated, and the right tonsil was enlarged. On the second day of examination, there was a systolic bruit and an increase of the area of dulness. The joint affection rapidly subsided under drug treatment, with rest the heart became normal, and after recovery no evidence of organic mischief could be detected. The child had not had a previous attack, nor was she subject to sore throat. Her mother, however, was rheumatic, and had had several attacks of Rheumatic Fever.

The second case was that of a youth of nineteen years. He had had four or five previous attacks of Acute Rheumatism, varying in intensity. He developed a polyarthritis, and on examination presented an enlarged heart, with an evidently old-standing double aortic bruit. The heart became more enlarged, and a mitral systolic bruit developed. This remained after the attack of Rheumatic Fever had subsided, and the patient, who had previously been able to be about and carry on his work as a clerk, was now wholly confined to bed. The heart muscle was evidently permanently damaged, and compensation was never re-established.

Of these two cases of Rheumatism, the first was the more severe attack, but no cardiac damage resulted; whereas,in the second and less severe attack of Rheumatism, the heart, which was already damaged by several previous attacks, was again further injured and permanently damaged. In case number two, the previous attacks of Rheumatism had not been so well marked as regards the joints, which was an interesting point, as it helped to show that as the patient grew older the rheumatic attacks conformed more to the typical type.

Case three was that of a boy of eight years of age. The temperature was elevated, and the patient complained of slight sore throat and vague pains in the joints. He had frequently complained of similar symptoms, but these had usually passed off quickly, and the boy was apparently himself again in a day or two. On examination, the heart was slightly dilated, and a systolic bruit was present. After recovery from the rheumatic attack, the systolic bruit disappeared, but a presystolic murmur remained.

The last case which I shall mention was that of a boy of fourteen. He showed well-marked joint affection, but throughout the attack there was no characteristic heart symptom, and on recovery no cardiac damage.

The above cases, taken from daily general practice, serve to demonstrate what I have already said in a previous part of the paper regarding Acute Rheumatism, namely, that the gravity of the heart lesion does not depend on the severity of the attack, but that a mild attack may leave a permanently damaged heart, whereas a severe or moderately severe attack may leave no cardiac impairment; again, that successive mild attacks

in children are very liable to produce permanent damage to the heart, and that these same mild attacks are very liable to be overlooked and disregarded; that the mitral and aortic valves are the most frequent situations of endocarditic damage, and that attacks may become more severe, and conform more to the typical type, as the patient becomes older. That inflammation and enlargement of the tonsils, and sore throat, are present has also been shown by the cases above remarked on.

IV.

Treatment of Cases of Acute Rheumatism, and Preventive Means to be taken in those with Rheumatic Diathesis and Hereditary Tendency. Differential Diagnosis.

After the diagnosis of Acute Rheumatism, the most important subject is that of the treatment of the disease. And I now propose to give briefly the usual treatment, with a few remarks on the preventive means which might be taken in those with the Rheumatic Diathesis and hereditary tendency. In the treatment of Acute Rheumatism, one must always keep in mind the likelihood of subsequent heart lesions, and conduct the treatment with a view to combating this pernicious resultant as far as it lies in one's power to do so. The patient must be rested as much as possible and convalescence prolonged. For absolute rest, the patient must be kept in a comfortable bed and between blankets. The night clothes must be made of flannel, and so constructed that they can be readily changed (a most necessary point, on account of the sweating), and should open down the front, so as to allow the physician free access to the chest for examination of the heart. The room should be well ventilated and warm. The diet is the same as for other fevers, namely, principally milk, diluted with mineral water. If this is not well borne, broths and soups may be

given. As convalescence is established, the diet becomes more liberal, but must still remain light and mostly of the milk variety, red meats being prohibited. Plenty of liquid, in the form of water, barley-water or lemonade, should be given. For the joint condition, wrapping in cotton wool and supporting by splints is usually all ~~this~~ that is required. If the pain is very severe, local soothing lotions or blistering may be necessary. The drug treatment consists in giving one or other of the Salicylate group, namely, Sodium Salicylate, Salicin or Aspirin. Sodium Salicylate may be given to an adult in 15 grain doses every three hours, until the pain is relieved; then every four hours till the temperature subsides. It should then be given less frequently and in smaller doses, and then withdrawn. Dr. Lees recommends its being given in combination with Sodium Bicarbonate and being vigorously pushed. The Sodium Bicarbonate lessens the liability to acid intoxication, which is indicated by vomiting, acetonuria, drowsiness and air-hunger. He prescribes for adults 15 grains Sodium Salicylate, with 30 grains of Sodium Bicarbonate, every two hours from 6 a.m. to 10 p.m., and once during the night; for children between 7 and 12 years,

10 grains, and under this age 5 grains, at the same intervals. Salicin, introduced by Dr.Maclagan, is less depressing than Salicylate of Soda, and may be given in 20 grain doses every two hours until the pain is relieved, and thereafter reduced in the same manner as the Sodium Salicylate. It is considered to be the best drug for children. Aspirin, when used, should be given in 15 grain doses every two hours, in the same way as Sodium Salicylate. Like Salicin, it is especially useful in children. If these drugs are badly tolerated, or fail in their action, Oil of Wintergreen, 20 minims every two hours in milk, may be given, or Antipyrin or Antifebrin may be resorted to. The Salicylates relieve the pain, and appear to reduce the swelling of the joints. They do not, however, appear to exert any marked influence in preventing the cardiac lesion. Sodium Bicarbonate is extensively used alone by some, and is credited with all the specific properties of the salicylic group. In the giving of these drugs, it is most necessary that the bowels should be kept open, and a daily evacuation is essential. If the salicylates fail to reduce the temperature, quinine may be given, and if this fails, the usual measures, such as sponge and graduated hot

bath, should be resorted to. The intramuscular injection of a solution of Magnesium Sulphate has been advocated, and Dr. Algernon Jackson of Philadelphia publishes a paper giving in detail his method, and claiming most satisfactory results. He also applies a lotion of Magnesium Sulphate locally to the affected joints. Bee stings and injection of Formic Acid in the region of the affected joints have also been tried and successes claimed for them by their advocates. Vaccine and serum treatment has not yet been fully developed, and so far the results obtained from their use have not been very encouraging. They have, however, in all probability, a great future before them.

In persons convalescing from an attack of Rheumatism, in those who have had previous attacks, and in those with a Rheumatic diathesis and hereditary tendency, special precautions should be taken to prevent an attack of Rheumatism - warm clothing, careful attention to the diet,Hygienic surroundings, avoidance of getting wet, chilled or over-fatigued, careful attention to the hygiene of the mouth and nasopharynx, and avoidance of contact with cases of Rheumatism. In children, special

attention should be paid to sore throat, growing pains, and nervous symptoms, and these effectively and efficiently treated, and not allowed, as so often happens at present, to proceed to endocarditis and chorea before the medical man is called in.

The diagnosis of Acute Rheumatism in adults is as a rule comparatively easy. From Acute Gout it is distinguished by occurring in youth and affecting either sex, by affecting the larger joints, and wandering from joint to joint, and by the continuous fever; from Acute Rheumatoid Arthritis by the joints affected, and by the action of salicylates in relieving the symptoms; from Pyaemia by the absence of rigors, the wandering nature of the joint affections, and the absence of cerebral and spleen symptoms. In Gonorrhoeal Arthritis, the knees, tarsal and carpal joints are the ones affected. There is usually a gonorrhoeal history, and salicylates have no marked influence in controlling the condition. In Osteomyelitis, which may be wrongly diagnosed as Acute Rheumatism, the general symptoms are more marked, and as a rule the local symptoms are more severe. In infants, Acute Rheumatism, when it occurs, has to be diagnosed from Infantile Paralysis or Infantile Scurvy.

V.

Summary.

In this brief paper on Acute Rheumatism, firstly, an Historical Survey has been given, in which the etiology has been discussed, and the various theories of the many observers put forward. It has been shown how the older theories, embracing the Chemical and Cold theories, gave place to the microbic one; how, of the various microbic theories, that of Poynton and Paine fitted in most suitably with the nature, symptoms and pathology of the disease, and how those two observers have established conclusively the existence of the Diplococcus Rheumaticus as the causal organism in Rheumatic Fever. Again, it is pointed out that the tonsils are the entrance gates to the system for the Diplococcus, and that it reaches its various sites of selection by the lymph and blood streams. The special lesions and organs affected are then discussed at some length, and mention is made of the morbid changes occurring in these organs, and of the symptoms produced. The heart receives a good deal of attention, and the Rheumatic Nodule is described and discussed at some length. The predisposing causes and incidence are touched upon, and four cases occurring in daily

practice are brought forward as illustrative of points in previous parts of the paper. Next comes perhaps the most important part of the whole discussion, namely, the treatment, and the paper is concluded with a brief mention of preventive means to be taken in those predisposed to the disease, and a note on the Differential Diagnosis.
